Periscope

Miscellany

INTERNAL HYDROCEPHALUS. W. G. Spiller and A. R. Allen (Journal A. M. A., April 13).

The authors discuss the causes of internal hydrocephalus, reviewing the literature, and remark that while the occlusion of the aqueduct of Sylvius is not infrequently mentioned as an etiologic factor in the textbooks, it is rare to find references to actual cases of such occurence. They take the opportunity, therefore, to report a case in which the condition was induced by a probably congenital, almost total occlusion of the aqueduct. The subject was an elderly woman who was supposed to have been born hydrocephalic, but in spite of this and of an epilepsy that had lasted for years she had lived to the advanced age of 62, and retained her faculties fairly well developed. The aqueduct, while almost entirely occluded, was well lined throughout by a layer of ependymal cells which would not be the case had its occlusion been due to neuroglia proliferation, as in a previous case reported by Spiller. This, and the long continuance of the condition through the patient's whole life, indicates the congenital nature of the obstruction.

CEREBRAL LOCALIZATION AND THE STUDY OF PSYCHIATRY, WITH ESPECIAL REFERENCE TO THE MECHANISM OF HALLUCINATIONS AND ILLUSIONS AND THE CLASSIFICATION OF THE FORMS OF INSANITY. Chas. K. Mills, M.D. (The British Medical Journal, Sept. 29, 1906).

Coarse cerebral lesions when diffuse are effective in the production of hallucinations and illusions, but not if strictly focal, and it is evident therefore that the part played by the associative mechanism of the brain is most important in the symptomatology of insanity. The author's classification of insanities is into the teratological or abiotrophic and the pathological or acquired. Under the first would be classed congenital idiocy and imbecility, dementia præcox, dementia choreica and other presenile insanities, and senile dementia; also those less in degree, such as the melancholia-mania group, paranoia, hysteria, epilepsy and psychasthenia. The pathological insanities would include those due to diffuse destructive organic disease, as the forms of imbecility or mental defect due to postnatal causes, general paresis, syphilitic insanity, and traumatic insanity; also the insanities due to toxemias and exhaustion. C. D. CAMP.

PARALYSIS OF THE EYE MUSCLES FOLLOWING SPINAL ANESTHESIA. Alvin. Ach, M.D. (Muench. med. Woch., March 26, 1907).

The etiology of the paralysis of the eye muscles, following spinal anesthesia is still obscure. Adam, who observed the first case of paresis of the muscles, thought the manifestations were due to hemorrhage in the nucleus of the sixth nerve. Loeser considered it to be due to the toxic action of the drug on the nucleus or nerve involved. He points out the affinity and selective action that some poisons have for certain nerves and nerve centers, as lues, alcohol, diphtheria and leal. Landow agrees with Loeser. Lang thought it to be perpipheral neuritis, whereas Baisch, in his case of double-sided abducens paralysis, was under the impression that it was an apoplectic insult. Ach is of the opinion that it is not due to the toxic action of the drug on the nucleus in the floor of the fourth ventricle, and the anatomical relations make it nearly impossible for any solution to enter the fourth ventricle from below. His view is that the poisons act directly on the nerves. Paralysis of the eye muscles occured from four to eleven days after the operation and lasted from six to forty-three days in the case observed by him. He gives the following rules for spinal anesthesia: (1) The most harmless drug should be used. (2) Tropacocaine is better than stovain as the latter has a marked influence on the motor nerves. Use small doses. (3) Do not use concentrated solution. (4) Rest of patient after operation, with head and shoulders raised.

F. J. CONZELMANN (U. S. Army).

THE PATHOLOGY OF PARALYSIS AGITANS. C. D. Camp (Journal A. M. A., April 13).

Camp, after giving a review of the literature of the pathologic findings in the nervous system in paralysis agitans, reports the results of his examination of fourteen cases, in eight of which he was able to study the peripheral nerves and muscles, and in two the ductless glands also. The most constant lesion in the nerve centers was a fibrosis of the capillary blood vessel of the spinal cord, which by rendering them more prominent, caused them to appear more numerous. The posterior and lateral column regions seemed most involved. There was no degeneration of nerve fibers of the cord in any case, and in only two cases was this observed in the peripheral nerves with the Weigert hematoxylin stain; in two others there was a swelling of the myelin sheaths, accompanied in one case by swelling of the axis cylinders. There was considerable pigmentation of the Betz cells in the paracentral lobule, but in only two cases were distinctly degenerated cells observed. He discusses the various theories of the disease, and thinks that many of the pathologic conditions found, and to which the disorder has been attributed, are only coincident senile changes. The muscular theories are also discussed, and in nine cases in which he examined the muscles, one of them during life, he found pathologic changes. There was swelling of the muscle fibers, which were round in cross-section instead of polygonal, multiplication of nuclei, etc., his findings agreeing in the main with those of Schwenn, Schiefferdecker and Idelsohn, indicating, he is inclined to believe, a specific change in the muscles in paralysis agitans, though he admits that this is not yet definitely proven. Camp, in considering the pathogenesis of the affection, finds most reason to believe that it is due to a toxemia, and that this is connected with disordered parathyroid function. In the two cases in which he was able to examine the ductless glands he found the parathyroids in a decidedly pathologic condition; in both there was a peculiar fatty infiltration, especially in relation to the blood vessels. Such a finding, in connection with the experimental evidence as to the effect of parathyroidectomy and the therapeutic results of Berkley in the treatment of paralysis agitans with parathyroid extract, furnish, he thinks, strong proof that the parathyroids play an important part in the pathogenesis of paralysis agitans. His conclusions are given as follows: (1) Paralysis agitans is not a neurosis nor is it senility. (2) The anatomic basis of the symptoms, muscular rigidity, tremor and the symptoms dependant on them lies in the affection of the muscles. (3) The disease is probably a general toxemia, and there is evidence that it is due to alteration in the secretion of the parathyroid glands. The case histories of the patients of which Camp made the pathologic studies are appended to his article.